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EXAMINER	
HA, JULIE	

ART UNIT	PAPER NUMBER
1654	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/553,669	LEE ET AL.	
	Examiner	Art Unit	
	JULIE HA	1654	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *08 November 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *42-60* is/are pending in the application.

4a) Of the above claim(s) *48 and 57* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *42-47,49-56 and 58-60* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Amendment after Non-final rejection filed on November 6, 2008 is acknowledged. Claims 42-60 are pending in this application. Claims 48 and 57 remain withdrawn from further consideration as being drawn to nonelected species. Claims 42-47, 49-56 and 58-60 are examined on the merits in this office action.This application contains claims drawn to an invention nonelected with traverse in the reply filed on June 8, 2007. A complete reply to the final rejection must include cancellation of nonelected claims or other appropriate action (37 CFR 1.144) See MPEP § 821.01.

Withdrawn Objection and Rejection

1. Objection to claims 42 and 52 are hereby withdrawn in view of Applicant's amendment to the claims.

2. Rejection of claims 42-44 and 52-53 under 35 U.S.C. 102(b) as being anticipated by Eisenbach-Schwartz et al (US 2002/0072493 A1) is hereby withdrawn in view of Applicant's amendment and arguments.

Maintained Rejections

35 U.S.C. 112, 2nd

3. The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

4. Claims 42-47, 49-56 and 58-60 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 42 and 52 recite, “wherein said soluble Nogo receptor-1 polypeptide comprises an NT domain, eight leucine-rich repeats, and an LRRCT domain.” The phrase “eight leucine-rich repeats" is unclear. It is unclear what eight leucine-rich repeats are referring to. For example, it is unclear if the eight leucine-rich repeats means a sequence "LLLLLLLL" or if it means leucine-rich regions throughout the peptide sequence. Further, it is unclear how many leucine residues would be encompassed in leucine-rich repeats. It is unclear if there are 8 LRRs throughout the peptide or protein sequence or 8 leucine residues would encompass 1 leucine-rich region. According to www.ebi.ac.uk/interpro/IEntry?ac=IPR001611 (enclosed), “Leucine-rich repeats (LRR) consist of 2-45 motifs of 20-30 amino acids in length that generally folds into an arc or horseshoe shape. LRRs occur in proteins ranging from viruses to eukaryotes, and appear to provide a structural framework for the formation of protein-protein interactions” (see p. 1). The specification has not fully defined what is meant by 8 LRRs. The specification discloses that “Full-length Nogo receptor-1 consists of a signal sequence, an N-terminus region (NT), eight leucine-rich repeats (LRR), and a LRRCT region (a leucine-rich-repeat domain C-terminal of the eight leucine-rich repeats…" (see paragraph [0031]) and that “Aβ peptides-like other ligands of NgR—require the entire LRR region of the NgR protein for binding…” (see paragraph [0065]). However, the specification does not describe what 8 LRRs are and where these are located. Looking at SEQ ID NO:3 of instant application, it is unclear where the 8 leucine-rich repeats would be located in the sequence, since there are leucine residues throughout the protein sequence. Further, since it is unclear where the 8 leucine-rich repeats are

located in the sequence, it is also unclear where the Leucine-rich repeat domain C-terminal of eight leucine-rich repeats (LRRCT) domain is located. Because claims 43-47 and 49-51 depend from indefinite claim 42 and claims 53-56 and 58-60 depend from indefinite claim 52 and do not clarify the point of confusion, they must also be rejected under 35 U.S.C. 112, second paragraph.

Response to Applicant's Arguments

5. Applicant argues that "claims 42 and 52 require that the soluble Nogo receptor -1 polypeptide comprises an NT domain, eight leucine-rich repeats, and an LRRCT domain…The specification also discloses the polypeptide sequence of, e.g., human and rat Nogo receptor-1 (see id. at Table 1). Thus, the specification discloses the polypeptide sequence of human and rat Nogo receptor-1, that full-length Nogo receptor-1 has eight leucine-rich repeats, and that C-terminal to those eight leucine-rich repeats, there is an additional leucine rich repeat domain." Applicant further argues that "In addition, two references available at the time of the invention, PCT/US02/32007 and PCT/US03/25004, which were incorporated by reference in their entireties in the present specification, describe additional soluble Nogo receptor-1 polypeptides that may be used in the methods of the invention." Applicant further argues that "Current Opinion in Structural Biology 11: 725-32 (2001) (attached hereto as Exhibit A), provides a detailed structure-function review of leucine-rich repeats, including how to identify leucine-rich repeat consensus sequences (page 725)."

6. Applicant's arguments have been fully considered but have not been found persuasive. The instant specification discloses full length human and rat Nogo receptor-1 (see SEQ ID NOS: 1-2, Table 1). SEQ ID NOS: 1 and 2 comprise leucine residues throughout the protein sequence. It is still unclear where the 8 leucine-rich repeats are located in the sequence, and thus, it is unclear where the C-terminal portion to those eight leucine-rich repeats are located. Again, looking at SEQ ID NO:3 of instant application, it is unclear where the 8 leucine-rich repeats would be located in the sequence, since there are leucine residues throughout the protein sequence. For example, the Kobe reference (exhibit A) indicates that leucine-rich regions (LRR) have the conserved pattern LxxLxLXXN/CxL, wherein x is any amino acid, L is leucine, valine, isoleucine and phenylalanine, N/C is Asparagine or Cysteine residue (see p. 725, right column). Page 728 indicates consensus sequences of LRR (see bottom of Table 2). The Examiner has reviewed SEQ ID NO:3, looking for the LRR, as indicated by Kobe reference. However, the sequence has leucine residues throughout the protein sequence. For example, starting at around residue 79, there are leucine residues, phenylalanine residues, isoleucine residues and valine residues that may indicate LRR. It is unclear where the 8 leucine rich region is located in SEQ ID NO:3. Since the 8 leucine rich region is unclear, it is unclear where the C-terminal region of the 8 leucine rich region is located. The C-terminal region of 8 leucine rich region can be located around residue 107 (8 leucine rich region starting around 79 to 101). The 8 leucine rich region may be located around residue 107 and the C-terminal starting around residue 134, and so on. Therefore, it is unclear where the 8 leucine-rich repeats would be

located in the sequence, and therefore, it is unclear where the C-terminal domain (LRRCT) would be located.

In regards to the incorporation by reference, the MPEP states the following: "an incorporation by reference must be set forth in the specification and must: (1) Express a clear intent to incorporate by reference by using the root words "incorporate(e)" and "reference" (e.g., incorporate by reference"); and (c) Essential material may be incorporated by reference, but only by way of an incorporation by reference to a U.S. patent or U.S. patent application publication, which patent or patent application publication does not itself incorporate such essential material by reference. "Essential material" is material that is necessary to" (1) provide a written description of the claimed invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same, and set forth the best mode contemplated by the inventor of carrying out the invention as required by the first paragraph of 35 U.S.C. 112; (2) Describe the claimed invention in terms that particularly point out and distinctly claim the invention as required by the second paragraph of 35 U.S.C. 112…" (see MPEP 608.01(p)). This incorporation by reference is therefore, improper.

Rejection-35 U.S.C. 103

7. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

8. The factual inquiries set forth in *Graham* **v.** *John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966), that are applied for establishing a background for determining obviousness under 35 U.S.C. 103(a) are summarized as follows:

1. Determining the scope and contents of the prior art.
2. Ascertaining the differences between the prior art and the claims at issue.
3. Resolving the level of ordinary skill in the pertinent art.
4. Considering objective evidence present in the application indicating obviousness or nonobviousness.

9. This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

10. Claims 42-45, 47, 51-53 and 60 are rejected under 35 U.S.C. 103(a) as being unpatentable over Strittmatter (US Patent No. 7,119,165) in view of Strittmatter SM (J. Mol. Neurosci., 2002, 19(1/2): 117-121, filed with IDS, NPL33).

11. Strittmatter SM (US Patent No.) teaches the soluble NgR1 polypeptide having 83 amino acid residues (see SEQ ID NO: 55). Strittmatter teaches that the soluble NgR1 polypeptide is used for the treatment of central nervous system disease, disorder or

injury, and the term CNS includes, altered CNS function resulting from physical trauma to cerebral or spinal chord tissue, viral infection, autoimmune mechanism, genetic mutation and neurodegenerative diseases or disorders (see column 11, lines 29-35). Patent '165 further teaches that the typical dosage comprises 1 pg/kg to 100mg/kg body weight, preferred dosage for systemic administration comprise 100 ng/kg to 100 mg/kg; preferred dosages for direct administration to a site via microinfusion comprise 1 ng/kg to 1 μg/kg body weight (see column 24, lines 36-45). The difference between the reference and the instant claims is that the reference does not teach the reduction of β amyloid levels, specifically the treatment of Alzheimer's disease and the therapeutically effective amount is from 1 μg/kg to 10 mg/kg.

12. However, Strittmatter teaches in Alzheimer's Disease (AD), it is generally accepted that neuronal loss is initiated by the accumulation of the β-amyloid (Aβ) peptide and that this results in cognitive dysfunction (see Introduction). Further, Strittmatter teaches that "while the primary pathological event in AD is the loss of nerve cells, there are two major reasons for considering means of promoting axonal growth as a therapeutic approach for AD…if successful therapies are developed to delay or halt neuronal death in AD, then means to promote increased axonal growth and new synaptic connections from remaining cells should provide a mechanism for recovery of lost function as opposed to simply halting the progression of disease" (see p.117, Introduction, left column). Further, the Strittmatter teaches that the NgR protein contains a signal sequ4ence followed by 8 leucine-rich repeat (LRR) domains, a LRR carboxy-terminal cysteine-rich flanking domain, a unique region and a glycophosphatidylinositol

(GPI) anchorage site (see Figure 1, and p. 120, left column, 1st full paragraph). Additionally, Strittmatter teaches that the blockage of Nogo receptor pathway is a therapeutic target for the treatment of Alzheimer's disease (see abstract and p. 120, left column, last paragraph).

13. Therefore, it would have been obvious to one of ordinary skill in the art to combine the teachings of Strittmatter (US Patent No. 7,119,165) and Strittmatter to try the use of soluble Nogo-receptor polypeptide for treating neurodegenerative diseases involving b amyloids, such as Alzheimer's disease. US Patent No. '165 teaches that the soluble NgR-1 polypeptide is used in treatment of neurodegenerative diseases (SEQ ID NO: 55), and Strittmatter SM teaches that promoting axonal growth as a therapeutic approach for AD, and cites Nogo receptor pathway as a therapeutic target. One of ordinary skill in the art would have been motivated to combine the teachings with a reasonable expectation that since US Patent No. '165 teaches the treatment of neurodegenerative disease or disorders using the NgR-1 polypeptide (via decreasing Nogo-dependent inhibition of axonal growth in CNS neurons), and Strittmatter reference (J. Mol. Neurosci) discloses that promoting axonal growth as a therapeutic approach in AD. Further, since Strittmatter reference indicates that in AD, it is generally accepted that neuronal loss is initiated by the accumulation of the β-amyloid peptide and that this results in cognitive dysfunction, one would necessarily expect to reduce the levels of Aβ peptide in the mammalian brain when the soluble NgR-1 polypeptide is administered to the mammalian patient population. Furthermore, it would have been obvious to one of ordinary skill in the art to optimize the therapeutically effect amount of the NgR-1

polypeptide, since Patent No. '165 teach different preferred dosage amounts for different administration.

14. The MPEP states the following: Generally, differences in concentration or temperature will not support the patentability of subject matter encompassed by the prior art unless there is evidence indicating such concentration or temperature is critical. *"[W]here the general conditions of a claim are disclosed in the prior art, it is not inventive to discover the optimum or workable ranges by routine experimentation."* In re Aller, *220 F.2d 454, 456, 105 USPQ 233, 235 (CCPA 1955)* (Claimed process which was performed at a temperature between 40°C and 80°C and an acid concentration between 25% and 70% was held to be *prima facie* obvious over a reference process which differed from the claims only in that the reference process was performed at a temperature of 100°C and an acid concentration of 10%.); see also Peterson, 315 F.3d at 1330, 65 USPQ2d at 1382 ("*The normal desire of scientists or artisans to improve upon what is already generally known provides the motivation to determine where in a disclosed set of percentage ranges is the optimum combination of percentages.*"); In re Hoeschele, *406 F.2d 1403, 160 USPQ 809 (CCPA 1969)* (Claimed elastomeric polyurethanes which fell within the broad scope of the references were held to be unpatentable thereover because, among other reasons, there was no evidence of the criticality of the claimed ranges of molecular weight or molar proportions.). For more recent cases applying this principle, see Merck & Co. Inc. v. Biocraft Laboratories Inc., 874 F.2d 804, 10 USPQ2d 1843 (Fed. Cir.), cert. denied, 493 U.S. 975 (1989); In re Kulling, 897 F.2d 1147, 14 USPQ2d 1056 (Fed. Cir. 1990); and In re Geisler, 116 F.3d

1465, 43 USPQ2d 1362 (Fed. Cir. 1997). There is a motivation to optimize the dosage concentrations, since the normal desire of scientists or artisans want to improve upon what is already known, and the MPEP states that this *provides the motivation to determine where in a disclosed set of percentage ranges is the optimum combination of percentages* . There is a reasonable expectation of success, because routine optimization would at least arrive at the optimal dosage that is the most effective in treating the condition or disorders being treated. From the teachings of the references, it is apparent that one of the ordinary skills in the art would have had a reasonable expectation of success in producing the claimed invention. Thus, the invention as a whole is *prima facie* obvious over the references, especially in the absence of evidence to the contrary.

Response to Applicant's Arguments

15. Applicant argues that "the Examiner has failed to establish a *prima facie* case of obviousness…there is no apparent reason to combine the prior art reference cited by the Examiner to arrive at the claimed invention." Applicant further argues that "The '165 patent does not discloses polypeptide of Nogo receptor-1 wherein said polypeptide inhibit Nogo receptor-mediated neurite outgrowth inhibition…the '165 patent teaches a method of treating a central nervous system disease, disorder or injury, e.g., spinal cord injury." Applicant argues that "patent '165 does not teach or suggest a method of treating a disease, disorder, or condition associated with plaques of Aβ peptide in a mammalian brain, comprising administering a therapeutically effective amount of soluble Nogo receptor-1 polypeptide." Furthermore, Applicant argues that "these

deficiencies are not cured by the disclosure of Strittmatter…while Strittmatter mentions the generally accepted pathology of Alzheimer's Disease (AD), "that neuronal loss is initiated by the accumulation of the β-amyloid (Ab) peptide and that this results in cognitive dysfunction," Strittmatter contemplates the promotion of axonal growth as therapeutic approach, not reducing Aβ peptide." Applicant further argues that "the Examiner is basing her rejection on the present method claims on inherent obviousness."

16. Applicant's arguments have been fully considered but have not been found persuasive. Strittmatter patent teaches the soluble NgR1 polypeptide having 283 amino acid residues (see SEQ ID NO: 55) that is very similar to instant SEQ ID NO: 3. The difference between the two sequences is that SEQ ID NO:55 has 283 residues, missing the first Pro at N-terminus and Ala at position 285 at C-terminus. However, the residues 1-283 are comprised within SEQ ID NO:3 of instant application. Therefore, the reference teaches a soluble NgR1 polypeptide comprising an NT domain, eight leucine-rich repeats, and an LRRCT domain (at C-terminus to 8 leucine rich repeats). Strittmatter teaches that the soluble NgR1 polypeptide is used for the treatment of CNS disease, disorder or injury (including neurodegenerative diseases or disorders), as described in the body of the rejection, above. Strittmatter (J. Mol. Neurosci) teaches that in AD, it is generally accepted that neuronal loss is initiated by the accumulation of the β-amyloid peptide and that this results in cognitive dysfunction. As described above, Strittmatter teaches that "while the primary pathological event in AD is the loss of nerve cells, there are two major reasons for considering means of promoting axonal growth as

a therapeutic for AD…if successful therapies are developed to delay or halt neuronal death in AD, then means to promote increased axonal growth and new synaptic connections from remaining cells should provide a mechanism for recovery of lost function as opposed to simply halting the progression of disease." Strittmatter further asserts that the blockage of Nogo receptor pathway is a therapeutic target for the treatment of Alzheimer's disease.

Since Strittmatter patent teaches that NgR1 polypeptide is used for the treatment of CNS disease, disorder, or injury (including neurodegenerative diseases or disorders) and Strittmatter reference teaches that AD is neuronal loss initiated by the accumulation of the β-amyloid peptide that results in cognitive dysfunction, it would have been obvious to one of ordinary skill in the art to combine the teachings of the prior arts, since both treatment of central nervous system diseases, such as neurodegenerative diseases or disorders. Furthermore Strittmatter patent teaches the treatment of neurodegenerative disease or disorders using the NgR-1 polypeptide (via decreasing Nogo-dependent inhibition of axonal growth in CNS neurons), and Strittmatter reference discloses that promoting axonal growth as a therapeutic approach in AD.
One of ordinary skill in the art would have been motivated to combine since Strittmatter reference teaches that "if successful therapies are developed to delay or halt neuronal death in AD, then means to promote increased axonal growth and new synaptic connections from remaining cells should provide a mechanism for recovery of lost function…" NgR-1 is used for treatment of central nervous system disease, such as neurodegenerative diseases or disorder via decreasing Nogo-dependent inhibition of

axonal growth in CNS neurons. Strittmatter reference teaches that in AD, it is generally accepted that neuronal loss is initiated by the accumulation of the Aβ peptide and promoting axonal growth is a therapeutic approach for AD, administration of NgR-1 peptide for treatment of central nervous system disease would necessarily reduce the levels of Aβ peptide in a mammalian brain. Axonal growth would necessarily reduce the Aβ peptide levels in the brains of mammals being administered with NgR-1 polypeptide. The rejection was based on the *prima facie* obviousness of the combined prior arts. Administration of NgR-1 polypeptide would necessarily reduce the levels of the Aβ peptide, since this peptide accumulation is associated with neuronal loss, and promoting axonal growth would delay neuronal death in AD, implying reduced accumulation of Aβ peptide. Therefore, the combined prior arts is *prima facie* obvious over the instant claims.

Conclusion

17. **THIS ACTION IS MADE FINAL.** Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a). No claim is allowed.

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of

the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to JULIE HA whose telephone number is (571)272-5982. The examiner can normally be reached on Mon-Thurs, 5:30 AM to 4:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Cecilia Tsang can be reached on 571-272-0562. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Cecilia Tsang/
Supervisory Patent Examiner, Art Unit 1654

/J. H./
Examiner, Art Unit 1654